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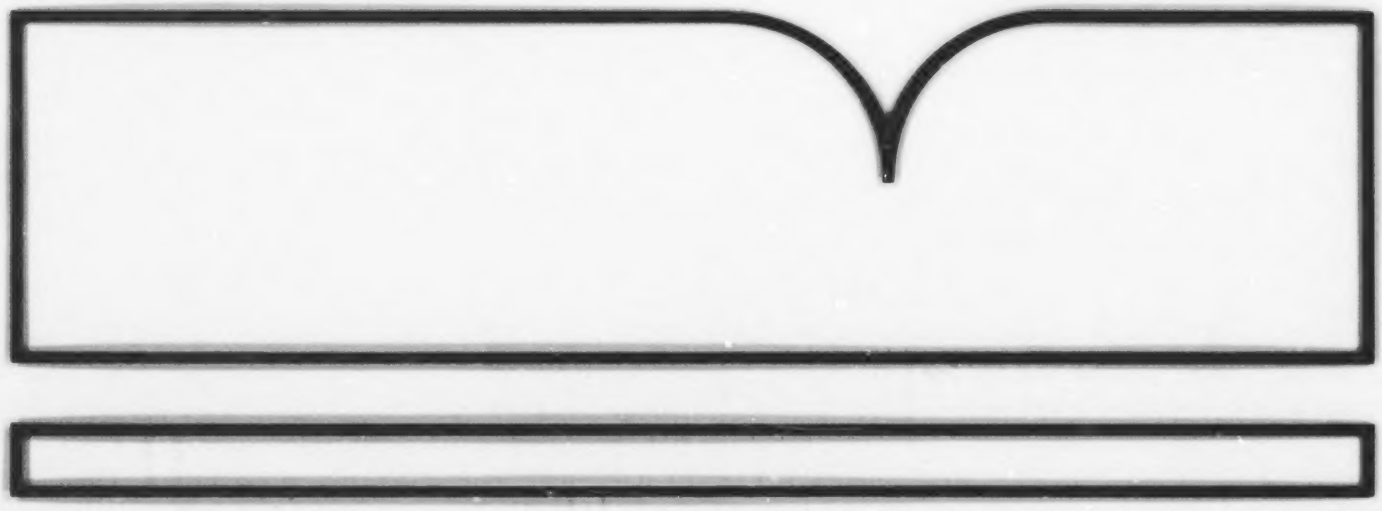

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BASIS FOR PRIMARY AIR QUALITY CRITERIA AND STANDARDS

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16. ABSTRACT

The Environmental Criteria and Assessment Office and the Office of Air Quality Planning and Standards are charged with responsibility for reviewing and assessing air quality criteria and air quality standards, respectively. Since adoption of the 1977 Clean Air Act Amendments, the U.S. Environmental Protection Agency has published criteria documents for all of the criteria pollutants and completed action on hydrocarbons, lead, and ozone standards. Currently ozone and lead documents are again under review, while proposals for carbon monoxide, nitrogen dioxide, and particulate matter ambient standards have been published. This paper summarizes both the basis for existing health criteria and standards and the status of current Agency review of the criteria and standards.

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BASIS FOR PRIMARY AIR QUALITY CRITERIA AND STANDARDS

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INTRODUCTION

The Environmental Criteria and Assessment Office (ECAO) and the Office of Air Quality Planning and Standards (OAQPS) are charged with responsibility for reviewing and assessing air quality criteria and air quality standards, respectively. Since adoption of the 1977 Clean Air Act Amendments, the U.S. Environmental Protection Agency (EPA) has published criteria documents for all of the criteria pollutants and completed action on hydrocarbons, lead, and ozone standards. Currently ozone and lead documents are again under review, while proposals for carbon monoxide, nitrogen dioxide, and particulate matter ambient standards have been published. This paper summarizes both the basis for existing health criteria and standards and the status of current Agency review of the criteria and standards.

Carbon Monoxide National Ambient Air Quality Standards

Background

On April 30, 1971, EPA promulgated in the Federal Register (36 FR 8186) national ambient air quality standards (NAAQS) for carbon monoxide (CO). The scientific, technical, and medical bases for the CO NAAQS are contained in Air Quality Criteria for Carbon Monoxide (AP-64) published by the U.S. Department of Health, Education, and Welfare in March 1970. Both primary (health based) and secondary (welfare based) standards were set at levels of 9 parts per million (ppm), 8-hour average, and 35 ppm, 1-hour average, neither to be exceeded more than once per year. The preamble to the regulation stated:

The national primary standard for carbon monoxide, proposed on January 30, 1971, was based on evidence that low levels of carboxyhemoglobin in human blood may be associated with impairment of ability to discriminate time intervals. In the comments, serious questions were raised about the soundness of this evidence. Extensive consideration was given to this matter. The conclusions reached were that the evidence regarding impaired time-interval discrimination had not been refuted and that less restrictive national standard for carbon monoxide would therefore not provide the margin of safety which may be needed to protect the health of persons especially sensitive to the effects of elevated carboxyhemoglobin levels above 2 percent. It is the Administrator's judgment that attainment of the national standards for carbon monoxide will provide an adequate safety margin for protection of public health and will protect against known and aniticipated adverse effects on public welfare.

Assessment of New Health Effects Evidence

The evidence cited as the basis of the original NAAQS for CO derives from work done by Beard and Wertheim regarding impairment of ability to discriminate time intervals. As the result of the failure of several researchers to replicate the Beard and Wertheim results at 2 percent carboxyhemoglobin (COHb), the implications and use of this evidence for standard setting have changed since the original CO standards were promulgated.

New evidence of health effects from CO exposure has been published since 1971, as evaluated in the revised Criteria Document for CO (EPA-600/8-79-022) and in a 1979 OAQPS Staff Paper. In assessing new CO research, EPA identified cardiovascular system effects as one of several types of effects attributed to CO. Citing the work of Anderson et al. (1973) and Aronow and Isbell (1973), EPA stated that aggravation of angina pectoris by CO is an adverse health effect for at least two major reasons. First, the angina reflects hypoxia which may also result in cardiovascular damage that is unquantifiable, using present technology. Second, aggravation of angina may be the first in a series of progressively more serious symptoms that accompany cardiovascular disease. Thus, consistent with the advice of the Clean Air Scientific Advisory Committee (CASAC), EPA concluded that CO exposures reulting in COHb levels of 2.7 to 2.9 percent were linked to adverse health effects in some portion of the estimated five to seven million persons with angina pectoris in the U.S. population.

Other health effects associated with exposure to CO include: (1) central nervous system (CNS) effects (Rockwell and Weir, 1975; Putz et al., 1976; Putz et al., 1979); (2) pulmonary function and exercise effects (Horvath et al., 1975; Raven et al., 1974) and (3) perinatal effects (Longo, 1977; Peterson,

1981; Hoppenbrouwers et al., 1981). Although several investigators report CNS effects and reduced exercise capacity in humans, EPA and CASAC did not clearly identify these effects as being adverse. Much of the information available on fetal effects of CO is from animal toxicology studies, and it remains very difficult to extrapolate these effects from animals to humans due to uncertainty about differences in sensitivity and uptake of CO between various animal species and humans.

Current Status of Review of the Standards

In accordance with Sections 108 and 109 of the Clean Air Act as amended, EPA reviewed and revised criteria upon which to base primary and secondary NAAQS for CO. The revised Criteria Document was published on August 18, 1980, simultaneously with the Federal Register notice of proposed rulemaking (45 FR 55066). As a result of the review and revision of criteria, EPA proposed to retain the existing primary 8-hour standard at 9 ppm and to lower the primary 1-hour standard to 25 ppm. The FR notice also proposed revocation of the secondary standards for CO, because welfare effects have been observed only at relatively high CO concentrations that rarely, if ever, occur in the ambient air. In addition, the proposal included a change from deterministic to statistical standards (i.e., allowable exceedances would be stated as expected values and not explicit values). Finally, EPA proposed to use daily interpretation for exceedance of the CO standards, so that exceedances would be determined on the number of days on which the 1-hour or 8-hour average levels are above the standard levels.

Developments in early 1983 caused EPA to question the validity of research by Dr. Aronow and have forced an extension of the CO standard review. EPA has assessed the impact on the CO standards of the diminished credibility of the Aronow studies, as evaluated in an Addendum to the 1979 CO Criteria Document and CO staff paper (Review of the NAAQS for Carbon Monoxide: Reassessment of Scientific and Technical Information), both of which were reviewed by CASAC in September, 1983. Based on this review, the CASAC supported Staff Paper recommendations that the 8-hour standard be set within the range of 9-12 ppm and the 1-hour standard be set within the range 25-35 ppm, and the Agency is still considering these recommendations along with other factors before proceeding with decisions on the CO NAAQS. In addition, EPA and other independent labora-

tories are conducting studies similar to the Aronow angina studies which should provide a markedly improved CO health effects data base in the near future.

Lead National Ambient Air Quality Standards

Background

On October 5, 1978, EPA published in the Federal Register (43 FR 46246) a national primary and secondary ambient air quality standard (NAAQS) for lead at a level of 1.5 micrograms lead per cubic meter of air (mg Pb/m^3), averaged over a calender quarter. This final rulemaking followed a 1976 court order to list lead as a criteria pollutant in accordance with provisions of Sections 108 and 109 of the Clean Air Act.

In establishing the level of the primary standard, EPA determined that young children (age 1-5 years) should be regarded as a group within the general population that is particularly sensitive to lead exposure. The lead NAAQS is based on preventing most (99.5 percent) children in the United States from exceeding a blood lead level of 30 micrograms per deciliter of blood (mg Pb/dl). Selection of 30 mg/dl as a "maximum safe" blood lead level was based on the following considerations:

(a) The maximum safe blood lead level should be somewhat lower than the threshold for lead-induced hemoglobin decrements (40 mg Pb/dl).

(b) The level should be well below the then-apparent threshold for nervous system effects (50 mg/dl).

(c) The maximum safe blood lead level need not be as low as the detection point for interference in heme biosynthesis, as indicated by elevated levels of erythrocyte protoporphyrin (EP).

(d) The level should be no higher than that level (30 mg Pb/dl) characterized in 1978 by the Center for Disease Control of the Public Health Service (and endorsed by the American Academy of Pediatrics) as reflecting undue pediatric exposure to lead.

Based on data used to determine the maximum safe blood lead level for an individual child, EPA took into account variations in individual susceptibilities and lead exposures in determining a maximum safe geometric mean blood lead level for the sensitive population as a whole. Given a lognormal population distribution of blood lead with a geometric standard deviation of 1.3, the

maximum safe geometric mean blood lead level for young children was determined to be 15 mg Pb/dl, in order to keep 99.5 percent of the pediatric population below the level of 30 mg Pb/dl.

EPA further calculated, based on then-available epidemiological data (Hammer et al., 1972; Angle et al., 1974; Goldsmith, 1974; Johnson et al., 1975), that of the target population mean blood lead level of 15 mg Pb/dl, 12 mg Pb/dl should be attributed to "non-air" lead sources unaffected by a lead NAAQS (43 FR 46246). That left a contribution of 3 mg Pb/dl to blood lead levels to be allowed from air sources. Studies of changes in blood lead levels with different air lead levels were then used to estimate an "air-lead to blood-lead ratio" of 1:2 (Griffin et al., 1975; Azar et al., 1975; Yankel et al., 1977). This means that a continuous exposure to 1.5 mg Pb/m^3 air would be expected to increase a child's blood lead level by 3 mg Pb/dl. Thus, a level of 1.5 mg/m^3 was selected for the lead NAAQS.

Based on a study showing increases in blood lead levels in adults after seven weeks of exposure (Griffin et al., 1975) and because of the greater risk of exposure of young children, a calendar monthly averaging time using the average of 24-hour measurements, was originally proposed by EPA (42 FR 63076). This averaging period was extended to a calendar quarter after consideration of public comments and because of technical and statistical problems that would be associated with a monthly standard based solely on one-in-six day sampling. Based on the relatively stable distribution of lead levels over a prolonged period and the relatively slow equilibration of blood lead levels in response to changes in air lead levels, the Agency concluded a calendar quarterly averaging time would improve the validity of air quality data gathered without significantly reducing the protectiveness of the standard.

Assessment of New Health Effects Evidence

Since promulgation of the NAAQS for lead in 1979, considerable improvements have occurred in the lead data base, especially in key areas listed below. More detailed information concerning each of these areas can be found in sections of the current draft criteria document (CD) specified below in parenthesis. It is important to note that this draft document is in the process of peer review by CASAC as part of the present NAAQS review. Consequently, only a preliminary assessment of the new information as it relates to the current review is

possible at this time. Completion of the CD is expected in late 1985. Included among major issues and important findings evaluated in the CD are:

(a) The extent of lead exposure in U.S. populations and important demographic covariates influencing such exposures. Recent surveys demonstrate that a significant portion of young children in the U.S. have blood lead levels in excess of 30 mg Pb/dl, and blood lead levels tend to be higher among urban non-white children in lower income families (CD, 11.3.3).

(b) Relationships between external lead exposure and absorbed lead. Several new analyses have attempted to quantify the impact of airborne lead, either directly through inhalation, or indirectly via soil, dust, or the foodchain, on children's blood lead levels. No assessments of simultaneous exposures to all media have been done (CD, 7.3; 11.3.4; 11.4).

(c) Relative contributions of various lead sources in the environment to total absorbed exposure. In addition to the previously mentioned analyses, there have been new studies utilizing analyses of stable lead isotope ratios to determine sources (e.g., gasoline, paint, soil, dust, food) of lead in blood. The results of these studies indicate that air lead, either directly via inhalation or indirectly via ingestion, contributes significantly to total human body burdens of lead (CD, 11.4-11.5) and that gasoline lead usuage is an important, key source of airborne lead contributing to exposures of U.S. population groups.

(d) Health effects associated with low-level lead exposures of children. Small, but significant deficits in IQ, school performance, and social behavior have been associated in children with blood lead levels as low, and possibly lower, than the 40 mg/dl threshold level for hemoglobin decrements. Other types of neurotoxic effects, such as altered electrophysiological brain wave patterns in children (as well as altered peripheral nerve conduction velocities in adults), have also been reported at lower levels. The medical significance of these latter findings, as well as results of the neurobehavioral studies, remain to be fully defined. They are, however, generally consistent with low-level lead-induced alterations in several neurotransmitter systems and in neural subcellular functions, such as mitochondrial respiration and protein synthesis (CD, 12.4). Disruption of vitamin-D metabolism and other indications of widespread impacts of lead-induced interference with synthesis of heme in many organ systems have also been found at blood lead levels that range to well below 30 mg/dl.

(e) Health effects associated with exposures of adults, such as lead-induced blood pressure increases. Recently published epidemiology studies show associations between increases in blood pressure in adult males and blood lead levels down to as low as 7-30 mg/dl.

The draft criteria document concludes that substantial impetus is provided for revising the "maximum safe" blood lead level downward to a level below 30 mg/dl (CD, p. 1-144), based on: (1) the new neuropsychologic and electrophysiological findings; (2) evidence of low-level lead effects on other functional

systems (e.g., cellular energetics, globin synthesis, vitamin D metabolism); and (3) animal toxicological studies showing a long persistence of lead-induced neurobehavioral alterations well into adulthood after termination of perinatal lead exposure, and that no threshold may exist for certain lead-induced neurochemical effects.

Current Status of Review of the Standard

A draft staff paper has been prepared by OAQPS outlining the rationale for possible revision of the lead NAAQS, based on the draft Criteria Document. The staff paper, reviewed by CASAC in May, 1985, will be revised to reflect the final Criteria Document and will be used as part of a decision package on the lead NAAQS to be reviewed internally and by CASAC in 1986.

Nitrogen Dioxide National Ambient Air Quality Standards

Background

On April 30, 1971, the Environmental Protection Agency (EPA) published in the Federal Register (36 FR 8186) primary and secondary national ambient air quality standards (NAAQS) for nitrogen dioxide (NO_2). Identical primary and secondary standards for NO_2 were set at 0.053 ppm (100 mg/m^3), averaged over one year. The scientific and medical bases for these standards are contained in the criteria document, "Air Quality Criteria for Nitrogen Oxides," published by EPA in January 1971 (AP-84). The primary standard set in 1971 was mainly based on a group of epidemiology studies (Shy et al., 1970a,b; Pearlman et al., 1971) conducted in Chattanooga, which reported respiratory effects in children exposed to low level NO_2 concentrations over long-term periods. Reevaluation of the Chattanooga studies based on more recent information (especailly regarding the accuracy of the air quality monitoring method for NO_2 used in the studies) indicates that these studies provide only limited evidence for an association between health effects and ambient exposures to NO_2. However, other new information summarized below, confirms the need for maintaining NO_2 ambient standards.

In accordance with provision of Sections 108 and 109 of the Clean Air Act as amended, EPA Has been reviewing the need for new or revised NO_2 standards since September 1977. In addition to reviewing the existing annual NO_2 standard, the Administrator is required to promulgate a short-term (1 to 3 hours)

NO_2 primary standard unless it is determined that there is no significant evidence that such a standard is required to protect public health.

A revised Criteria Document for NO_x, reviewed by CASAC in 1978-1979 was completed in late 1981 and, based on the Criteria Document (1982a), OAQPS prepared a paper, "Review of the National Ambient Air Quality Standards for Nitrogen Dioxide: Assessment of Scientific and Techncal Information (OAQPS Staff Paper)", which evaluated the available scientific and technical information most relevant to the review of the NO_2 NAAQS. The OAQPS Staff Paper (1982b) also presented recommendations on alternative approaches to revising the standards.

Assessment of New Health Effects Evidence

A variety of respiratory system effects have been reported to be associated with exposure to NO_2 concentrations less than 2.0 ppm in humans and animals. The most frequent and significant NO_2-induced respiratory effects reported in the scientific literature at the time the Criteria Document and OAQPS Staff Paper were published include (1) altered lung function and symptomatic effects observed in controlled human exposure studies and in community epidemiological studies, (2) increased prevalence of acute respiratory illness and symptoms observed in outdoor community epidemiological studies and in indoor community epidemiclogical studies comparing residents of gas and electric stove homes, and (3) lung tissue damage and increased susceptibility to infection observed in animal toxicology studies. As the Criteria Document concludes, results from these several kinds of studies collectively provide evidence indicating that certain human health effects may occur as a result of exposures to NO_2 concentrations at or approaching recorded ambient NO_2 levels.

At the time of proposal, based on controlled human exposure studies, EPA concluded that human pulmonary function effects of clear health concern resulting from single, short-term exposures of less than 3 hours duration have been unambiguously demonstrated only at concentrations (greater than 1.0 ppm) well in excess of ambient exposure levels typically encountered by the public. More subtle health effects that were of uncertain health significance, such as mild symptomatic effects, had been reported for some asthmatics after a single 2-hour exposure to 0.5 ppm.

The principal evidence reviewed in the OAQPS Staff Paper and proposal on the effects of repeated short-term exposures came from a series of

cross-sectional epidemiological (community) studies, some ongoing, which reported increased prevalence of acute respiratory illness and impaired lung function in children living in homes with gas stoves (a source of NO_2) as compared to children living in electric stove homes. Findings from several animal studies demonstrating reduced resistance to infection due to NO_2 exposures support the belief that NO_2 exposures are probably related to the effects observed in these indoor epidemiological studies. Estimation of NO_2 concentrations potentially associated with the reported health effects was especially difficult because the investigators did not measure short-term NO_2 concentrations in the homes of the subjects in the indoor epidemiology studies. Based on NO_2 monitoring data from other gas stove homes, it was estimated that the health effects observed in gas stove homes, if due to NO_2 exposure, were likely to be associated with frequent, repeated short-term peak exposures to NO_2 levels ranging up to 0.5 to 1.0 ppm and possibly as low as 0.15 to 0.30 ppm.

While the animal toxicology literature does not permit estimation of human effect levels at this time, it does document a variety of health effects from acute, chronic, and chronic with repeated peak exposures to NO_2. Findings from animal studies, e.g., emphysematous alterations in the lung (Port et al., 1977), other morphological changes in the lung (Aranyi et al., 1976), and increased susceptibility to infection (Ehrlich and Henry, 1968; Ito, 1971), involving chronic exposures to 0.5 ppm NO_2 or greater or chronic exposures to 0.1 ppm with repeated peaks of 1.0 ppm NO_2 suggest that chronic exposures to NO_2 may lead to serious adverse health effects in humans. While such exposure levels cannot be directly extrapolated to humans, given the similarities between man and animals, it is likely that the above types of effects observed in several animal species also occur in man, albeit at unknown exposure levels. These effects may include development of chronic respiratory diseases and increased incidence of acute respiratory infection or disease. Less severe and generally reversible effects, e.g., biochemical changes (Menzel et al., 1977; Sherwin et al., 1974), interference with hormone metabolism (Menzel, 1980), and possible interference with liver metabolism (Miller et al., 1980), have been reported in animals exposed once to NO_2 concentrations in the range 0.2-0.5 ppm.

Since proposal, EPA's ECAO has reviewed the scientific studies that have become available since CASAC closure on the Criteria Document and OAQPS Staff Paper and that were identified by EPA staff and/or in public comments on the NO_2

proposal. The principal points from ECAO's review of the new studies are summarized below.

(1) The more recent controlled human exposure studies (most of which are presently in unpublished form) present mixed and apparently contradictory results concerning respiratory effects in asthmatics and healthy individuals at concentrations in the range of 0.1 to 4.0 ppm NO_2. Some new studies have reported an increased effect on airway resistance or lung function when challenged by a bronchoconstricting agent and NO_2 (Ahmed et al., 1982; Bauer et al., 1984) while other recent studies have reported no statistically significant effects from NO_2 alone or with a bronchoconstricting agent (Hazucha et al., 1983; Kleinman et al., 1983; Ahmed et al., 1983). Only Kagawa and Tsuru (1979) have reported results possibly suggestive of short-term NO_2 effects on pulmonary function without combined provocative challenge by other agents (e.g., carbachol or cold air) for a group of 6 subjects exposed to 0.15 ppm NO_2. However, the small size of the decrements reported (all less than 5 percent) and questions regarding the statistical analyses used suggest caution in accepting the reported findings as demonstrating NO_2 effects on pulmonary function at 0.15 ppm. Further research by other investigators at exposure levels in the 0.1 to 0.5 ppm NO_2 range is underway and should provide valuable clarifying information on low-level NO_2 pulmonary function effects.

(2) The most recent indoor epidemiological studies by the British and Harvard groups indicate somewhat weaker findings of an association between NO_2 and respiratory effects than the original studies conducted by these groups cited in the Criteria Document and proposal notice. For example, an estimated odds ratio for respiratory illness before age 2 of 1.23 (p F 0.01) previously reported by the Harvard group (Speizer et al., 1980) has been reduced to 1.12 (p = 0.07) by the inclusion in the statistical analyses of data from additional children enrolled in the study (Ware et al., 1984). The association between residence in a gas stove home and respiratory illness before age 2 is no longer considered statistically significant. Nonetheless, the Ware et al. study continued to find small statistically significant decreases in pulmonary function when the data for this large sample of children were analyzed, although possible confounding by parental education may overstate this relationship. Some other indoor epidemiological studies (with much smaller statistical power) involving electric and gas stove homes have reported statistically significant increased rates of symptoms and illness in residents of gas stove homes

(Comstock et al., 1981; Helsing et al., 1982; Lebowitz et al., 1982), while other studies have failed to find any statistically significant associations with gas stove usage (Jones et al., 1982; Melia et al., 1982; Melia et al., 1983). Unfortunately, none of the recent studies have provided an assessment of short-term NO_2 levels in the residences of the subjects evaluated. Overall, then, the newly available data from indoor epidemiological studies do not appear to resolve the mixed results reported in earlier studies and do not provide much additional evidence substantiating or strengthening earlier conclusions regarding health effects potentially associated with repeated short-term exposures to NO_2 in gas stove homes.

(3) The results from the more recent animal studies further substantiate the NO_2 effects on immune function and increased susceptibility to infection. However, the lack of an acceptable method at this time for quantitative extrapolation of the animal data to man greatly limits their usefulness beyond providing qualitative support for analogous effects plausibly being associated with repeated short-term, high-level and chronic exposure to NO_2.

Current Status of Review of the Standards

Based on the data presented in the Criteria Document, analyses summarized in the OAQPS Staff Paper, and CASAC's recommendations, the Agency has concluded that the current 0.053 ppm NO_2 annual average standard adequately protects against adverse health effects associated with long-term exposures and provides some measure of protection against possible short-term health effects. The Agency is, therefore, currently considering retaining the existing annual average NO_2 standard, while continuing to evaluate the evidence bearing on whether a separate short-term standard is requisite to protect public health (49 FR 6866).

Ozone National Ambient Air Quality Standards

Background

On April 30, 1971, the Environmental Protection Agency (EPA) published in the Federal Register (36 FR 8186) primary and secondary national ambient air quality standards (NAAQS) for photochemical oxidants. Both standards were set at an hourly average level of 0.08 ppm not to be exceeded more than once per

year. Epidemiological research on asthmatics by Schoettlin and Landau (1961) was cited as the main basis for the primary standard.

In accordance with Sections 108 and 109 of the Clean Air Act as amended, EPA reviewed and revised the criteria upon which the original photochemical oxidants NAAQS were based. Based on the resulting Ozone Criteria Document (1978), the final rulemaking for revising these standards was published on February 8, 1979 (44 FR 8202), including the following changes: (1) raising the primary and secondary standards to 0.12 ppm, (2) changing the chemical designation of the standards from photochemical oxidants to ozone (O_3), (3) changing to a standard with a statistical rather than deterministic form, and (4) changing the definition of the point at which the standard is attained to "when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm is equal to or less than one."

Several factors were cited as a basis for setting the primary standard for O_3. Among these were: (1) the adverse health effect threshold could not be identified with certainty, (2) O_3 is a pulmonary irritant that affects respiratory mucous membranes as well as other lung tissue and impairs respiratory function at levels as low as 0.15 ppm for exercising persons (Delucia and Adams, 1977), (3) elevated numbers of asthma attacks when peak hourly oxidant concentrations reach about 0.25 ppm (Schoettlin and Landau, 1961), (4) increased susceptibility to bacterial infection in laboratory animals exposed to O_3 plus a bacterial challenge, (5) premature aging symptoms reported in rabbits (Stokinger, 1965), and (6) apparent synergistic effects on pulmonary function from exposure to 0.37 ppm O_3 and 0.37 ppm sulfur dioxide. The above information led EPA to conclude that a primary standard of 0.12 ppm (one-hour average) not to be exceeded more than one day per year on average would protect public health with an adequate margin of safety.

Since promulgation of the NAAQS for O_3 in 1979, a much improved data base has developed demonstrating health effects from short-term exposures to O_3. These effects range from unquantifiable symptomatic effects to the carefully measured pulmonary function decrements reported in clinical and laboratory field studies. In addition, animal data suggest potentially more serious long-term exposure effects, such as morphological and functional changes in the lung and alterations in body chemistry. These effects, as well as welfare effects, are under review in a revised criteria document released in July 1984 as a first external review draft.

A number of important controlled studies have reported significant decrements in pulmonary function associated with O_3 exposure. Results from studies of at-rest exposures to O_3 have demonstrated decrements in forced expiratory volumes and flows occurring at and above 980 $\mu g/m^3$ (0.5 ppm) of O_3 (Folinsbee et al., 1978; Horvath et al., 1979) Airway resistance is not clearly affected at these O_3 concentrations. At or below 588 $\mu g/m^3$ (0.3 ppm) of O_3, changes in pulmonary function do not occur during at rest exposure (Folinsbee et al., 1978), but the occurrence of some O_3 pulmonary symptoms has been suggested (König et al., 1980).

With moderate intermittent exercise at a V_E of 30 to 50 L/min, decrements in forced expiratory volumes and flows have been observed at and above 588 $\mu g/m^3$ (0.30 ppm) of O_3 (Folinsbee et al., 1978). With heavy intermittent exercise V_E = 65 L/min), pulmonary symptoms are present and decrements in forced expiratory volumes and flows are suggested to occur following 2-hr exposures to 235 $\mu g/m^3$ (0.12 ppm) of O_3 (McDonnell et al., 1983). Symptoms are present and decrements in forced expiratory volumes and flows definitely occur at 353 to 470 $\mu g/m^3$ (0.18 to 0.24 ppm) of O_3 following 1 hr of continuous very heavy exercise at a V_E of 80 to 90 L/min (Adams and Schelegle, 1983; Folinsbee et al., 1984) and following 2 hr of intermittent very heavy exercise at a V_E of 65 L/min (McDonnell et al., 1978; McDonnell et al., 1983). Increased f_R and decreased V_T, while maintaining the same V_E, occur with prolonged heavy ecercise when exposed at 392 to 470 $\mu g/m^3$ (0.20 to 0.24 ppm) of O_3 (McDonnell et al., 1983; Adams and Schelegle, 1983). While an increase in RV has been reported to result from exposure to 1470 $\mu g/m^3$ (0.75 ppm) of O_3 (Hazucha et al., 1973), changes in RV have not been observed following exposures to 980 $\mu g/m^3$ (0.50 ppm) of O_3 of less, even with heavy exercise (Folinsbee et al., 1978). Decreases in TLC and IC have been observed to result from exposures to 980 $\mu g/m^3$ (0.50 ppm) of O_3 of less, with moderate and heavy exercise (Folinsbee et al., 1978).

A close association has been observed between the occurrence of respiratory symptoms and changes in pulmonary function in response to acute exposure to O_3. This association holds for both the time-course and magnitude of effects. In studies of repeated exposure to O_3, symptoms have generally increased while pulmonary function has diminished on the second to third day of exposure, followed by partial or complete reversal of these findings on the fourth or fifth day. Insofar as cough and chest pain or irritation may interfere with the maximal inspiratory or expiratory efforts required to perform spirometric

maneuvers, such associations between symptoms and function might be expected. To date, epidemiological studies of the health effects of photochemical air pollution have not been designed specifically to test the comparative frequency or magnitude of response of symptoms versus functional changes. Which type of effect is more likely to occur within the polluted community is uncertain.

The symptoms found in association with clinical exposure to O_3 alone and with exposure to photochemical air pollution are similar but not identical. Eye irritation, one of the commonest complaints associated with photochemical pollution, is not characteristic of clinical exposures to O_3, even at concentrations of the gas several times higher than any likely to be encountered in ambient air. Other components of photochemical air pollution, such as aldehydes and PAN, are held to be chiefly responsible (National Air Pollution Control Association, 1970; Altshuller, 1977; National Research Council, 1977; U.S. Environmental Protection Agency, 1978; Okawada et al., 1979).

There is also evidence to suggest that for a specified concentration of O_3 other symptoms, as well, are more likely to occur in populations exposed to ambient air pollution than in subjects exposed in chamber studies, especially if O_3 is the sole pollutant administered in the chamber studies. The symptoms may be indicative of either upper or lower respiratory tract irritation. In epidemiological studies, cough has been reported at 0.08 ppm O_3 (Zagraniski et al., 1979) and at 0.10 ppm oxidants (Hammer et al., 1974; Makino and Mizoguchi, 1975). Some subjects have experienced cough during clinical exposure to 0.12 ppm O_3 (McDonnell et al., 1983). Nose and throat irritation have been reported in community studies in association with 0.10 ppm oxidants (Okawada et al., 1979; Makino and Mizoguchi, 1975), but not at or below 0.15 ppm O_3 in laboratory studies.

Between 0.15 and 0.30 ppm O_3, a variety of both respiratory and non-respiratory symptoms have been reported in controlled exposures. They include throat dryness, difficulty or pain in inspiring deeply, chest tightness, substernal soreness or pain, wheezing, lassitude, malaise, and nausea (DeLucia and Adams, 1977; Kagawa and Tsuru, 1979b; McDonnell et al., 1983). Most "symptom scores" have been positive at concentrations of 0.2 ppm O_3 and above; only two studies reported finding no symptoms at 0.3 ppm O_3 (Bennett, 1962; von Nieding et al., 1977). Symptoms tend to remit within hours after exposure is ended. Relatively few subjects have reported persistence of symptoms beyond 24 hours.

There are several possible explanations for the apparent differences in symptomatic effects between epidemiological and controlled human studies. They include differences in subject populations (e.g., children were subjects in two of the epidemiological studies but have not been studied clinically); factors affecting the perception of symptoms in one type of study compared to the other; or differences in the methods used to assess symptoms. Alternatively, the presence of highly reactive chemical species in polluted ambient air might be chiefly responsible for the symptoms or might interact synergistically with O_3 to initiate the symptoms.

Symptoms have commonly been assessed by the use of recording sheets combined with reliance on the subject's recall, usually right after exposure but sometimes several hours or days after exposure (e.g., community studies). While it is difficult to score the intensity of symptoms with confidence, the results obtained immediately after exposure have been noteworthy for their general consistency across studies. Moreover, as noted earlier, the association between changes in symptoms and objective functional tests has been impressive. Symptoms are therefore considered as useful adjuncts in assessing the effects of O_3 and photochemical pollution, particularly if combined with objective measurements.

Community epidemiological studies investigate "real world" exposure conditions but are usually limited by uncertainty due to numerous confounding variables. Effects reported to occur at maximum hourly concentrations of O_3 or oxidant in the ambient air ranging from 0.08 ppm to 0.17 ppm include: increased symptoms of respiratory irritation (Hammer et al., 1974; Zagraniski et al., 1979); decreased pulmonary function (Linn et al., 1980, 1983; Avol et al., 1983; Lebowitz at al., 1982; Kagawa 1982; Lippman et al., 1982); and aggravation of asthma (Whittemore and Korn, 1980). Although the above effects were reported to be associated with O_3/O_x concentrations at or below 0.17 ppm, these epidemiology studies are being critically assessed to determine if they can provide useful quantitative information on exposure-effect relationships.

Animal toxicology research has provided evidence for numerous adverse effects from O_3 exposure which would be impossible to obtain from human subjects. Many of these animal studies have involved long-term exposures and the use of invasive techniques. Effects reported in the literature include: morphological (structural) changes in the lung, such as damage to ciliated and Type I cells, hyperplasia of nonciliated bronchiolar and Type II cells,

inflammation, and thickening of interalveolar septa with continued exposure; pulmonary function changes, such as rapid and shallow breathing, enhanced airway reactivity, and evidence for distal airway narrowing with continued exposure; biochemical changes in the lung, such as increased antioxidant and cellular enzymes and increased collagen synthesis; effects on host defense mechanisms, such as increased susceptibility to bacterial infection, increased number and decreased function of alveolar macrophages; CNS effects; hematological effects; chromosomal, reproductive, and teratological effects; and effects on the liver and endocrine system. Most of these effects have been observed in various animal species following 0.1 to 1.0 ppm O_3 exposures lasting for periods ranging from several days to several months. The major difficulties of using animal data directly, however, are the uncertainty associated with dosimetry and species sensitivity differences between humans and animals and uncertainties regarding the biological or biomedical significance of the observed effects. EPA's Office of Air Quality Planning and Standards and Office of Research and Development are currently supporting development and application of dosimetry models which will improve utilization of animal data during air standard development by reducing uncertainty in extrapolating from animal to human effects.

Current Status of Review of Criteria and Standards

ECAO is currently revising the ozone criteria document based upon CASAC and public comments provided on a First External Review Draft prior to and at a CASAC meeting held in Springfield, Virginia on March 4-6, 1985. A draft staff paper, based on the revised criteria document and comments made at the CASAC meeting, is being prepared for public release in August 1985. Following further CASAC review, the criteria document and staff paper are expected to be finalized and ready to use in early 1986 for decision making purposes regarding the O_3 NAAQS.

Particulate Matter
National Ambient Air Quality Standards

Background

On April 30, 1971, the Environmental Protection Agency (EPA) published in

the Federal Register (36 FR 8186) primary and secondary national ambient air quality standards (NAAQS) for particulate matter. The primary standards (measured as total suspended particulate matter or "TSP") are 260 mg/m^3 annual geometric mean. The secondary standard (also measured as TSP) is 150 mg/m^3, averaged over a period of 24 hours, and not to be exceeded more than once per year.

The 1971 Federal Register notice did not provide a detailed description of the rationale for the standards. Nevertheless, it is possible to identify the most relevant support by comparing the standards with the final conclusions of the 1969 criteria document. Similarly, the rationale used at the time for selection of hi-volume samplers (which measure TSP, as opposed to other particle sampling instruments) was not explicitly defined in the Federal Register notice.

In accordance with provisions of Sections 108 and 109 of the Clean Air Act as amended, EPA has reviewed and revised the criteria upon which the existing primary and secondary standards are based, as evaluated in the new Criteria Document for Particulate Matter and Sulfur Oxides (EPA-600/8-82-029c). Also, the 1982 "Review of the National Ambient Air Quality Standards for Particulate Matter: Assessment of Scientific and Technical Information--OAQPS Staff Paper," available in EPA's regional libraries, summarizes the most relevant information contained in the revised criteria document that EPA staff believe should be considered in revisions of the particulate matter standards.

More than any other pollutant, "particulate matter" represents a broad class of chemically and physically diverse substances. Particulate matter exists as discrete particles in condensed (liquid or solid) phases ranging in size from molecular clusters of 0.005 mm to coarse particles on the order of 100 mm. Fine particles (F2.5 mm) typically originate by condensation of materials produced during combustion (e.g., inorganic lead) or atmospheric transformation (e.g., sulfuric acid). Coarse particles (J2.5 mm) are largely derived from mechanical processes such as grinding or wind erosion. Because fine and coarse particles are generally derived from different mechanisms, the chemical species comprising each mode are not usually evenly distributed within the biomodel distribution formed by the fine and coarse modes, respectively. The variable composition and physical properties of particles often affect health and welfare in different ways and should be considered when evaluating particle effects.

Assessment of New Health Effects Evidence

Information developed since the promulgation of the existing primary standards makes it clear that TSP is not the most appropriate indicator for particulate matter because it includes substantial quantities of larger particles (up to 25-45 mm) of less concern to health. New data on the bimodal distribution of airborne particles, respiratory tract deposition, and health effects suggest that a new size-specific indicator that includes particles less than or equal to a nominal 10 mm (PM_{10}) would be more appropriate. This indicator would ensure that the full range of particles penetrating to the sensitive alveolar region of the respiratory tract is included, and follows tracheobronchial penetration patterns in a somewhat conservative fashion during oronasal or mouth breathing. It places substantially greater emphasis on controlling smaller particles than TSP, but does not completely exclude larger particles from control.

The revised criteria document found that a number of the community epidemiological studies used to support the existing primary standards, including Winkelstein (1967), which appears to have provided major support for the level and form of the existing annual standard, were unsuitable for quantitative standard setting purposes. As a result, only a small number of short- and long-term epidemiological studies were cited as being useful in reaching conclusions regarding concentrations at which particulate matter is likely to produce health effects. The short-term (24-hour) exposure studies include a series of studies and reanalyses of daily mortality in London (Martin and Bradley, 1960; Martin, 1964; Ware et al., 1981; Mazumdar et al., 1981) and studies of bronchitis patients in London (Lawther et al., 1970). The long-term (annual) exposure studies examined changes in respiratory disease and lung function in children (Lunn et al., 1967), increases in respiratory disease symptoms and reductions in lung functions in adults (Ferris et al., 1973, 1976), and increases in respiratory symptoms in adults (Bouhuys et al., 1973). The staff assessment of these "quantitative" studies is discussed in the OAQPS Staff Paper (1982).

Results from controlled human, toxicological, animal, and qualitative epidemiological studies cited in the revised criteria also provide useful information on health effects and other senstivie subgroups of the population (e.g., children) not addressed in the more "quantitative" studies. Such information is particularly useful in evaluating margins of safety associated with alternative standard levels.

Current Status of Review of Standards

On March 20, 1984, EPA proposed revisions to the particulate matter standards (49 FR 10408). These revisions include:

(1) That TSP as an indicator for particulate matter be replaced for both of the primary standards by a new indicator that includes only those particles with an aerodynamic diameter smaller than or equal to a nominal 10 micrometers (PM_{10});

(2) That the level of the 24-hour primary standard be changed to a value to be selected from a range of 150 to 250 mg/m^3 and that the current deterministic form of the standard be replaced with a statistical form that permits one expected exceedance of the standard level per year;

(3) That the level and form of the annual primary standard be changed to a value to be selected from a range of 50 to 65 mm/m^3, expressed as an expected annual arithmetic mean; and

(4) That the current 24-hour secondary TSP standard be replaced by an annual TSP standard selected from a range of 70 to 90 mg/m^3, expected annual arithmetic mean.

The Agency is still in the process of considering public comments and other imputs regarding the above proposed revisions in the PM NAAQS.

Sulfur Oxides
National Ambient Air Quality Standards

Background

On April 30, 1971, the Environmental Protection Agency (EPA) published the existing primary national ambient air quality standards (NAAQS) for sulfur oxides (36 FR 8186). Although both a 3-hour and annual secondary standard were published in 1971, a 1973 Federal Register notice announced the revocation of the annual secondary standard and reaffirmation of the 3-hour secondary standard (38 FR 25678). The primary standards, measured as sulfur dioxide (SO_2), are 0.14 ppm (365 mg/m^3), averaged over a period of 24 hours and not to be exceeded more than once per year, and 0.03 ppm (80 mg/m^3), annual arithmetic mean. The secondary standard also measured as SO_2, is 0.5 ppm (1300 mg/m^3) averaged over 3 hours, not to be exceeded more than once per year.

No detailed descriptions were provided in the Federal Register notice for rationales underlying the standards published in 1971. Nevertheless, the most

relevant support can be identified by comparing the standards with final conclusions in the 1970 criteria document. The 1973 proposal and promulgation notices were the first notices on NAAQS that provided specific rationales. Subsequent notices for NAAQS actions provided rationales explaining the basis for proposed NAAQS changes.

In accordance with provisions of Sections 108 and 109 of the Clean Air Act as amended, EPA reviewed and revised the criteria upon which the existing primary and secondary standards are based, as evaluated in the new criteria document for PM/SO_x (EPA-600/8-82-029a). Another document "Review of the National Ambient Air Quality Standards for Sulfur Oxides: Assessment of Scientific and Technical Information--OAQPS Staff Paper," available in EPA's regional libraries, summarizes the most relevant information that should be considered in the review of the sulfur oxide standards. As a result of the recently completed review of the scientific basis for the sulur oxides standards, several findings have developed that are different from those used to support the original standards. Concisely highlighted below are recent EPA findings particularly relevant to this standard review and a brief description of the basis for supporting various standard options.

Assessment of New Health Effects Evidence

A number of new studies have been performed since the promulgation of the original standards. New information from controlled human exposure studies demonstrates a clear response of sensitive individuals (asthmatics, atopics) to peak SO_2 exposures. Because such exposures can occur in the immediate vicinity of certain point sources, this informtion suggests that a standard to protect against peak exposures should be considered. The criteria review indicated that there are few new epidemiological studies available and that many of the older epidemiological studies previously considered to be quantitative should only be used qualitatively. The discussion of recent results from the review of the scientific basis of the sulfur oxides standards is organized according to averaging time (i.e., peak, 24-hour, and annual averages).

A number of recent controlled human exposure studies now demonstrate significant responses to SO_2 peaks below 1 ppm in sensitive individuals. In each case, responses below 1 ppm have only been reported to occur with moderate (or greater) levels of exercise. Caution must be used, therefore, in extending the results of these laboratory exposures to ambient conditions. In these

studies, (e.g., those by Sheppard et al., 1980; Kirkpatrick et al., 1982; etc.), asthmatics and "atopics" (individuals not diagnosed as asthmatic but with atopic disorders) exhibit changes in pulmonary mechanics, such as increases in airway resistance, that are indicative of significant bronchoconstriction after exposures of only 5 to 10 minutes of SO_2 concentrations even below 0.5 ppm (1300 mg/m^3). At or above 0.5 ppm, changes in functional measures are accompanied by perceptible symptoms such as wheezing, shortness of breath, and coughing. Available information indicates that these effects only occur with moderate (or greater) exercise levels and these sensitive individuals are about an order of magnitude more sensitive than "normal" subjects.

The major support for the existing 24-hour standard (set in 1971) is derived from studies conducted in London during the 1950's. Comparison of those studies that provided the basis for the original standards with the recently completed criteria review indicates that a number of studies are no longer considered reliable for quantitative purposes. Nevertheless, studies and reanalyses by Martin and Bradley (1960), Martin (1964), Mazumdar et al. (1981), and Ware et al. (1981) of London mortality data demonstrate increased risk of mortality as SO_2 levels approach or exceed 500 mg/m^3.

Unlike results of the 24-hour epidemiological studies, which indicate that some older studies can still be considered quantitatively reliable, none of the older long-term studies are considered to be quantitative. Although the criteria review found existing data on effects of long-term SO_2 exposure to be inconclusive and uncertain, the possibility of effects from continuous low-level exposure to SO_2 cannot be ruled out. Given the lack of quantitative epidemiological data suggesting long-term effects of SO_2 at or near the level of the current annual standard, no rigorous rationale can be presented to support a specific range of interest for an annual standard. Nevertheless, air quality analyses suggest the need to retain an annual NAAQS to prevent increases in annual SO_2 levels in heavily populated urban areas with attendant possible effects due to large increases in population exposures.

Current Status of Review of the Standards

The standard review schedule currently calls for proposal of the sulfur oxides standards in fall, 1985, with promulgation to follow within a year.

Availability of Criteria Documents and Staff Papers

CO:

Air Quality Criteria for Carbon Monoxide (October 1979)
EPA-600/8-79-022
NTIS* - PB-81-244840

Revised Evaluation of Health Effects Associated with Carbon Monoxide Exposure (August 1984)
EPA-600/8-83-033F

Review of the NAAQS for Carbon Monoxide: Reassessment of Scientific and Technical Information (July 1984)
EPA-450/5-84-004

HC:

Review of Criteria for Vapor-Phase Hydrocarbons (August 1980)
NTIS - PB-82-136516

Pb:

Air Quality Criteria foe Lead (December 1977)
EPA-600/8-77-017
NTIS - PB-280411

Air Quality Criteria for Lead (October 1983)
EPA-600/8-83-028A

NO_x:

Air Quality Criteria for Oxide of Nitrogen (December 1982)
EPA-600/8-82-026F
PB-82-163337

Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technical Information

O_3:

Air Quality Criteria for Ozone and Other Photochemical Oxidants (April 1978)
EPA-600/8-78-004
NTIS - PB-80-124753

Air Quality Criteria for Ozone and Other Photochemical Oxidants (July 1984)
EPA-600/8-84-020A

PM and SO_x:

Air Quality Criteria for Particulate Matter and Sulfur Oxides (December 1981)
EPA-600/8-82-029F
NTIS - PB84-156777 (set)
PB84-156785-Vol. 1
PB84-156793-Vol. 2
PB84-156801-Vol. 3

Review of the National Ambient Air Quality Standards for Particulate Matter: Assessment of Scientific and Technical Information (OAQPS Staff Paper) (January 1982)
EPA-450/5-82-001
NTIS - PB-82-177874

Review of the National Ambient Air Quality Standards for Sulfur Oxides: Assessment of Scientific and Technical Information - OAQPS Staff Paper (November 1982)
EPA-450/5-82-007
NTIS - PB-84-102920

Technical Information
(OAQPS Staff Paper) (August 1982)
EPA-405/5-85-002
NTIS - PB--83-132829

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*National Technical Information Service, Department of Commerce, Springfield, Virginia 22161; Commercial: 703--487-4250; FTS: 737-4650.

**EPA, Center for Environmental Research Information, 26 West Saint Clair Street, Cincinnati, Ohio 45268; Phone: (513) 684-7562 (FTS is the same).